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L8	l1 and (transgen\$ or knockout)	51	L8
L7	L6 not l4	9	L7
L6	l1 and ER	12	L6
L5	L4 not l3	1	L5
L4	l1 and RXR	3	L4
L3	l1 and RAR	2	L3
L2	L1 near3 RAR	0	L2
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NEWS 30 Apr 11 Display formats in DGENE enhanced  
NEWS 31 Apr 14 MEDLINE Reload  
NEWS 32 Apr 17 Polymer searching in REGISTRY enhanced  
NEWS 33 Apr 21 Indexing from 1947 to 1958 being added to records in CA/CAPLUS  
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=> s Cre (3a) (fus? or ligat?)

L1 218 CRE (3A) (FUS? OR LIGAT?)

=> s l1 and (RXR or RAR or ER)

L2 37 L1 AND (RXR OR RAR OR ER)

=> s l2 and (transgen? or knockout)

L3 26 L2 AND (TRANSGEN? OR KNOCKOUT)

=> dup rem i3

PROCESSING COMPLETED FOR L3

L4 12 DUP REM L3 (14 DUPLICATES REMOVED)

=> d bib abs 1-  
YOU HAVE REQUESTED DATA FROM 12 ANSWERS - CONTINUE? Y/N(y)

L4 ANSWER 1 OF 12 CAPLUS COPYRIGHT 2003 ACS  
AN 2002:511498 CAPLUS  
DN 137:42577  
TI A \*\*\*transgenic\*\*\* mouse carrying a gene for a \*\*\*cre\*\*\* recombinase \*\*\*fusion\*\*\* protein regulated by synthetic estrogens  
IN Champon, Pierre; Metzger, Daniel  
PA Association pour le Developpement de la Recherche en Genetique Moleculaire  
Adergrem, Fr.  
SO Fr. Demande, 141 pp.  
CODEN: FRXXBL

DT Patent  
LA French  
FAN.CNT 1  
PATENT NO. KIND DATE APPLICATION NO. DATE

PI FR 2814642 A1 20020405 FR 2000-12570 20001003  
US 2002100098 A1 20020725 US 2001-853033 20010511  
WO 200208175 A2 20020411 WO 2001-182248 20010928  
WO 200208175 A3 20030109

W: CA, JP  
RV: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,  
PT, SE, TR

PRAI FR 2000-12570 A 20001003

US 2001-853033 A 20010511

AB A \*\*\*transgenic\*\*\* non-human metazoan, specifically a mouse, that carries the gene for a \*\*\*fusion\*\*\* protein of \*\*\*cre\*\*\* recombinase and a nuclear estrogen receptor is described. The receptor is responsive to synthetic estrogens, such as tamoxifen, but not to natural estrogens and so can be used to regulate recombination via loxP sites, e.g. at different developmental stages, allowing the anal. of the role of a gene at these stages. Specifically, the method is used to investigate the function of retinoid X receptor alpha. (\*\*RXR\*\*\* alpha.). The construction of the genes for \*\*\*RXR\*\*\* alpha. contg. loxP sites and the \*\*\*Cre\*\*\* recombinase \*\*\*fusion\*\*\* protein with an estrogen receptor is described. The fusion protein gene was placed under the control of tissue-specific promoters to limit the deletions to specific tissues. \*\*\*Transgenic\*\*\* mice carrying these genes were constructed by std. methods. Inactivation of the \*\*\*RXR\*\*\* alpha. gene in the epidermis resulted in alopecia and the development of cysts on the skin within 6-12 wk of administration of tamoxifen. The skin continued to degenerate and after 20 wk small wound-like lesions appeared. Inactivation of the \*\*\*RXR\*\*\* alpha. gene in adipocytes appeared to be without phenotype.

L4 ANSWER 2 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1

AN 2002:310382 BIOSIS

DN PREV20020310382

TI Ligand-dependent genetic recombination in fibroblasts: A potentially powerful technique for investigating gene function in fibrosis.

AU Zheng, Bing; Zhang, Zhaoping; Black, Carol M.; de Crombrugge, Benoit; Denton, Christopher P. (1)

CS (1) Center for Rheumatology, University College London, Rowland Hill St., Royal Free Campus, London, NW3 2PF; c.denton@rfc.ucl.ac.uk UK

SO American Journal of Pathology, (May, 2002) Vol. 160, No. 5, pp. 1609-1617.

http://ajp.amjpathol.org/.print.

ISSN: 0002-9440.

DT Article

LA English

AB Strategies for conditional induction of \*\*\*transgene\*\*\* expression in mice are likely to be valuable for testing the role of candidate genes in disease pathogenesis. We have developed a system for lineage-specific, ligand-dependent, induction of sustained \*\*\*transgene\*\*\* expression in fibroblastic cells in mice using a chimeric gene encoding the \*\*\*Cre\*\*\* - \*\*\*ER\*\*\* (T) \*\*\*fusion\*\*\* protein, under the control of a fibroblast-specific regulatory sequence from the proalpha2(I)collagen gene. Cre- \*\*\*ER\*\*\* (T) operates as a tamoxifen-dependent DNA recombinase to excise fragments flanked by specific LoxP consensus sequences. To test efficiency and ligand dependency of this strategy, Cre- \*\*\*ER\*\*\* (T)-expressing mice were backcrossed with heterozygous

ROSA26-LacZ reporter mice, in which a floxed-STOP cassette has been introduced upstream of a bacterial beta-galactosidase (LacZ) reporter gene at a ubiquitously expressed locus. Constitutive or tamoxifen-induced LacZ expression was examined in embryonic, neonatal, and adult compound-

\*\*\*transgenic\*\*\* mice. When pregnant ROSA26-LacZ females received a single dose of tamoxifen, high-level expression of LacZ in the skin was demonstrable from 24 hours after injection in double- \*\*\*transgenic\*\*\* embryos harboring both the Cre- \*\*\*ER\*\*\* (T) \*\*\*transgene\*\*\* and the target ROSA26-LacZ allele. High-level expression of LacZ was also induced postnatally by tamoxifen specifically in dermal and visceral fibroblasts. By allowing efficient embryonic or postnatal modification of alleles that have been targeted to incorporate LoxP sites, or to switch on

\*\*\*transgenes\*\*\* cloned downstream of the floxed-STOP cassette, this system will allow fibroblast-specific genetic perturbations to be induced at predetermined embryonic or postnatal time points. This should greatly assist in vivo functional studies of candidate genes in fibrotic diseases such as systemic sclerosis.

L4 ANSWER 3 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2

AN 2002:288147 BIOSIS

DN PREV2002020298147

TI Efficient recombination in diverse tissues by a tamoxifen-inducible form of Cre: A tool for temporally regulated gene activation/inactivation in the mouse.

AU Hayashi, Shigemi; McMahon, Andrew P. (1)

CS (1) Department of Molecular and Cellular Biology, Harvard University, 16 Divinity Avenue, Cambridge, MA, 02138; amcmahon@mcb.harvard.edu USA

SO Developmental Biology, (April 15, 2002) Vol. 244, No. 2, pp. 305-318.  
http://www.academicpress.com/db/.print.

ISSN: 0012-1606.

DT Article

LA English

AB In recent years, the Cre integrase from bacteriophage P1 has become an essential tool for conditional gene activation and/or inactivation in mouse. In an earlier report, we described a \*\*\*fusion\*\*\* protein between \*\*\*Cre\*\*\* and a mutated form of the ligand binding domain of the estrogen receptor (Cre-ERTM) that renders Cre activity tamoxifen (TM)

inducible, allowing for conditional modification of gene activity in the mammalian neural tube *in utero*. In the current work, we have generated a "transgenic" mouse line in which Cre-ERTM is ubiquitously expressed to permit temporally regulated Cre-mediated recombination in diverse tissues of the mouse at embryonic and adult stages. We demonstrate that a single, intraperitoneal injection of TM into a pregnant mouse at 8.5 days postcoitum leads to detectable recombination in the developing embryo within 6 h of injection and efficient recombination of a reporter gene in derivatives of all three germ layers within 24 h of injection. In addition, by varying the dose of TM injected, the percentage of cells undergoing a recombination event in the embryo can be controlled. Dose-dependent excision induced by TM was also possible in diverse tissues in the adult mouse, including the central nervous system, and in cultured cells derived from the "transgenic" mouse line. This inducible Cre system will be a broadly useful tool to modulate gene activity in mouse embryos, adults, and culture systems where temporal control is an important consideration.

L4 ANSWER 4 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
3

AN 2003:35107 BIOSIS

DN PREV200300035107

TI \*\*\*ER\*\*\* -based double iCre fusion protein allows partial recombination in forebrain.

AU Casanova, Emilio (1); Fehsefeld, Sandra; Lemberger, Thomas; Shimshek, Derya R.; Sprengel, Rolf; Mantamadiotis, Theo  
CS (1) Abteilung Molekulärbiologie der Zelle I., Deutsches Krebsforschungszentrum (DKFZ), Im Neuenheimer Feld 280, D-69120, Heidelberg, Germany; e.casanova@dkfz.de Germany  
SO Genesis The Journal of Genetics and Development. (November 2002, 2002) Vol. 34, No. 3, pp. 208-214. print.  
ISSN: 1526-854X.

DT Article

LA English

AB Here we describe the generation of a new tamoxifen-inducible double \*\*\*Cre\*\*\* \*\*\*fusion\*\*\* protein generated by fusing two ERT2 domains onto both ends of the iCre recombinase (a codon improved \*\*\*Cre\*\*\* recombinase). This \*\*\*Cre\*\*\* \*\*\*fusion\*\*\* protein (ERiCreER) had a twofold increased activity in cell culture assays than the previously described MerCreMer \*\*\*Cre\*\*\* double \*\*\*fusion\*\*\* protein. ERiCreER was targeted to the brain by placing it under the control of the promoter from the CamKIIalpha gene using a 170 kb BAC. The fusion protein was detected in hippocampus, cortex, striatum, thalamus, and hypothalamus but not in cerebellum. The ERiCreER was cytoplasmic in the absence of tamoxifen and translocated into the nucleus upon tamoxifen administration. The activity of the ERiCreER was tested *in vivo* by mating the CamKIIalpha ERiCreER "transgenic" line with mice harbouring exon 10 of the CREB gene flanked by two LoxP sites. In the absence of tamoxifen, no background activity was detected in mice older than 6 months. After tamoxifen administration, most if not all of the ERiCreER fusion protein translocated from the cytoplasm to the nucleus; however, only 5-10% of the "fixed" CREB allele was recombined. Recombination was also visualised at the cellular level by following the upregulation of the CREM protein, which corresponds precisely with CREB loss/recombination. Unlike in other tissues (Sohai et al., 2001; Tannah-Louet et al., 2002), it appears that in brain, although ERiCreER can bind tamoxifen, the Cre-recombinase cannot be fully activated.

L4 ANSWER 5 OF 12 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

AN 2002311838 EMBASE

TI Temporal Cre-mediated recombination exclusively in endothelial cells using Tie2 regulatory elements.

AU Forde A.; Consten L.; Grone H.-J.; Hammerling G.; Arnold B.  
CS B. Arnold, Department of Molecular Immunology, Division of Tumor Immunology, German Cancer Research Center, Heidelberg 69120, Germany.  
b.arnold@dkfz-heidelberg.de

SO Genesis, (2002) 33/4 (191-197).

Refs: 21

ISSN: 1526-854X CODEN: GNESFY

CY United States

DT Journal/ Article

FS 022 Human Genetics

029 Clinical Biochemistry

LA English

SL English

AB The versatility of the bacteriophage Cre/LoxP system is dependent on the availability of a spectrum of tissue-specific Cre "transgenic" mice to address a host of biological questions. In this paper, we report on the generation of an inducible Tie2Cre "transgenic" mouse line that facilitates gene targeting exclusively in endothelial cells. The temporal manner of recombination is feasible through the use of a \*\*\*Cre\*\*\* -estrogen receptor \*\*\*fusion\*\*\* protein \*\*\*ER\*\*\* (T2) and was, in practical terms, achieved by feeding the animals the estrogen antagonist tamoxifen orally for 5 weeks. High efficiency of recombination was found in the vast majority of endothelial cell populations examined, as monitored by an EGFP reporter mouse line. Critically, no EGFP expression was observed in any uninduced mice. This inducible Cre line will be a very beneficial asset to investigating the role of endothelial specific genes in the adult mouse and to induce "transgenes" in the endothelium in an extremely efficient manner. .COPYRG. 2002 Wiley-Liss, Inc.

L4 ANSWER 6 OF 12 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

AN 2001099876 EMBASE

TI An efficient system for conditional gene expression in embryonic stem cells and in their *in vitro* and *in vivo* differentiated derivatives.

AU Valier L.; Mandip J.; Markossian S.; Lukaszewicz A.; Dehay C.; Metzger D.; Chambon P.; Samant J.; Savatier P.  
CS P. Savatier, Lab. de Biol. Moléculaire/Cellulaire, Ctr. Natl. de la Rech. Scientifique, Inst. Natl. Rech. Agronomique LA913, 46 Allée d'Italie, 69364 Lyon Cedex 07, France. Pierre.Savatier@ens-lyon.fr

SO Proceedings of the National Academy of Sciences of the United States of America, (27 Feb 2001) 98/5 (2467-2472).

Refs: 32

ISSN: 0027-8424 CODEN: PNAS6

CY United States

DT Journal/ Article

FS 004 Microbiology

021 Developmental Biology and Teratology

037 Drug Literature Index

LA English

SL English

AB We have developed a universally applicable system for conditional gene expression in embryonic stem (ES) cells that relies on tamoxifen-dependent Cre recombinase-loxP site-mediated recombination and bicistronic gene-trap expression vectors that allow "transgene" expression from endogenous cellular promoters. Two vectors were introduced into the genome of recipient ES cells, successively: (i) a bicistronic gene-trap vector encoding the beta-galactosidase/neo(R) "fusion" protein and the \*\*\*Cre\*\*\* - \*\*\*ER\*\*\* (T2) ( \*\*\*Cre\*\*\* recombinase "fused" to a mutated ligand-binding domain of the human estrogen receptor) or (ii) a bicistronic gene-trap vector encoding the hygro(R) protein and the human alkaline phosphatase (hAP), the expression of which is prevented by tandemly repeated stop-of-transcription sequences flanked by loxP sites. In selected clones, hAP expression was shown to be regulated accurately by 4-hydroxy-tamoxifen. Strict hormone-dependent expression of hAP was achieved (i) *in vitro* in undifferentiated ES cells and embryoid bodies, (ii) *in vivo* in virtually all the tissues of the 10-day-old chimeric fetus (after injection of 4-hydroxy-tamoxifen to foster mothers), and (iii) *ex vivo* in primary embryonic fibroblasts isolated from chimeric fetuses. Therefore, this approach can be applied to drive conditional expression of virtually any "transgene" in a large variety of cell types, both *in vitro* and *in vivo*.

L4 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2003 ACS  
AN 2001:47713 CAPLUS

DN 135:147951

TI Impaired adipogenesis and lipolysis in the mouse upon selective ablation of the retinoid X receptor alpha, mediated by a tamoxifen-inducible chimeric Cre recombinase (Cre-ERT2) in adipocytes

AU Imai, Takeshi; Jiang, Ming; Chamson, Pierre; Metzger, Daniel

CS Institut du Génétique et de Biologie Moléculaire et Cellulaire, Centre National de la Recherche Scientifique/Institut National de la Santé et de la Recherche Médicale/Université Louis Pasteur, Collège de France, Illkirch, 67404, Fr.

SO Proceedings of the National Academy of Sciences of the United States of America (2001), 98(1), 224-228  
CODEN: PNAS6; ISSN: 0027-8424

PB National Academy of Sciences

DT Journal

LA English

AB Retinoid X receptor alpha, ( \*\*\*RXR\*\*\* .alpha.) is involved in multiple signaling pathways, as a heterodimeric partner of several nuclear receptors. To investigate its function in energy homeostasis, the authors have selectively ablated the \*\*\*RXR\*\*\* .alpha. gene in adipocytes of 4-wk-old "transgenic" mice by using the tamoxifen-inducible Cre-ERT2 recombination system. Mice lacking \*\*\*RXR\*\*\* .alpha. in adipocytes were resistant to dietary and chem. induced obesity and impaired in fasting-induced lipolysis. Our results also indicate that \*\*\*RXR\*\*\* .alpha. is involved in adipocyte differentiation. Thus, the data demonstrate the feasibility of adipocyte-selective temporally controlled gene engineering and reveal a central role of \*\*\*RXR\*\*\* .alpha. in adipogenesis, probably as a heterodimeric partner for peroxisome proliferator-activated receptor gamma.

RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 8 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
4

AN 2001:271557 BIOSIS

DN PREV200100271557

TI Site- and time-specific gene targeting in the mouse.

AU Metzger, Daniel; Chambon, Pierre (1)

CS (1) Collège de France, Institut de Génétique et de Biologie Moléculaire et Cellulaire, CNRS/INSERM/ULP, 67404, Illkirch Cedex, C.U. de Strasbourg France

SO Methods (Orlando), (May, 2001) Vol. 24, No. 1, pp. 71-80. print.  
ISSN: 1046-2023.

DT Article

LA English

SL English

AB The efficient introduction of somatic mutations in a given gene, at a given time, in a specific cell type, will facilitate studies of gene function and the generation of animal models for human diseases. We have established a conditional site-specific recombination system in mice using a new version of the Cre/loxP system. The \*\*\*Cre\*\*\* recombinase has been "fused" to a mutated ligand binding domain of the human estrogen receptor ( \*\*\*ER\*\*\* ), resulting in a tamoxifen-dependent Cre recombinase, Cre-ERT, that is activated by tamoxifen, but not by estradiol. \*\*\*Transgenic\*\*\* mice were generated expressing Cre-ERT under the control of a cytomegalovirus promoter. Administration of tamoxifen to these \*\*\*transgenic\*\*\* mice induced excision of a chromosomally integrated gene flanked by loxP sites in a number of tissues, whereas no excision could be detected in untreated animals. However, the efficiency of excision varied between tissues, and the highest level (apprx40%) was obtained in the skin. To determine the efficiency of excision mediated by Cre-ERT in a given cell type, Cre-ERT-expressing mice were crossed with reporter mice in which expression of Escherichia coli beta-galactosidase can be induced through Cre-mediated recombination. The efficiency and kinetics of this recombination were analyzed at the cellular level in the epidermis of 6- to 8-week-old double \*\*\*transgenic\*\*\* mice. Site-specific excision occurred within a few days of tamoxifen treatment in essentially all epidermis cells expressing Cre-ERT. These results indicate that cell-specific expression of Cre-ERT in \*\*\*transgenic\*\*\* mice can be used for efficient tamoxifen-dependent Cre-mediated recombination at loci containing loxP sites, to generate site-specific somatic mutations in a spatiotemporally controlled manner. This conditional site-specific recombination system should allow the analysis of \*\*\*knockout\*\*\* phenotypes that cannot be addressed by conventional gene targeting.

L4 ANSWER 9 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
5

AN 2000:55232 BIOSIS

DN PREV200000055232

TI Temporally-controlled site-specific mutagenesis in the basal layer of the epidermis: Comparison of the recombinase activity of the tamoxifen-inducible Cre-ERT and Cre-ERT2 recombinases.

AU Indra, Arup Kumar; Warot, Xavier; Brocard, Jacques; Bonnet, Jean-Marc; Xiao, Jia-Hao; Chambon, Pierre (1); Metzger, Daniel

CS (1) Collège de France, Institut de Génétique et de Biologie Moléculaire et Cellulaire, CNRS/INSERM/ULP, 67404, Illkirch Cedex, C.U. de Strasbourg France

SO Nucleic Acids Research, (Nov. 15, 1999) Vol. 27, No. 22, pp. 4324-4327.





results in an increase in liver peroxisome number, marked hepatomegaly and induction of several genes encoding peroxisomal and other microsomal and mitochondrial enzymes involved in fatty acid metabolism. Chronic treatment of rodents with PP results in hepatocellular carcinoma. Species differences in PP responses have been found. For example, PP such as clofibrate and gemfibrozil, are highly effective lipid and cholesterol lowering drugs in humans but do not cause peroxisome proliferation and there is no evidence for increased liver cancers in patients receiving these drugs. A receptor, designated PP-activated receptor alpha (PPAR-alpha) is capable of trans-activating reporter genes containing a PP response (PPRE), but requires the presence of both PP, 9-cis retinoic acid and another receptor called RXR<sup>alpha</sup>. However, PP may not directly bind to PPAR-alpha but probably indirectly disturb cellular metabolism to liberate an endogenous ligand. Subsequent to the first identification of a PPAR-alpha, other members of this receptor family were found and designated PPAR-alpha, PPAR-beta (also called NUC1 and PPAR-delta) and PPAR-gamma. The alpha form is most abundant in liver and kidney, sites of peroxisome proliferation while the other two receptors are not significantly expressed in these tissues. On the basis of tissue-specific localization and spectrum of target gene activation, the physiological function of PPAR-alpha and PPAR-gamma appear to be related to fatty acid metabolism and regulation of adipogenesis. To gain insight into the function of PPAR-alpha and its role in the peroxisome proliferator response and hepatocellular carcinogenesis, gene targeting was used to develop a PPAR-alpha-deficient mouse. These animals are resistant to the pleiotropic effects of PP and no induction of any known target gene has been found. Recent studies on the phenotypes of these mice have led to an understanding of the mechanism of action of PP. They have also provided a useful model to establish the physiological role of PPAR-alpha in fatty acid homeostasis and inflammation.

L6 ANSWER 5 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 1997:346448 BIOSIS

DN PREV19979845651

TI Decreased expression of murine PPAR-gamma in adipose tissue during endotoxemia.

AU Hill, Molly R.; Young, Misty D.; McCurdy, Caren M.; Gimble, Jeffrey M.

CS Dep. Radiologic Technology, Univ. Okla. Health Sciences Cent., 801 NE 13th Street, Oklahoma City, OK 73190 USA

SO Endocrinology, (1997) Vol. 138, No. 7, pp. 3073-3076.

ISSN: 0013-7227.

DT Article

LA English

AB Infection-induced hyperlipidemia develops due to a combination of factors, one of which is decreased clearance of lipids from the bloodstream due to depressed synthesis of lipoprotein lipase (LPL). Recently, the peroxisome proliferator activated receptors (PPARs) have been shown to be important in the regulation of LPL, particularly PPAR-gamma. PPAR-gamma and its heterodimerization partner, RXR<sup>alpha</sup> have been shown to be transcriptional activators of LPL in co-transfection analysis. Therefore, we hypothesized that the decrease in LPL expression during endotoxemia may be a result of depressed PPAR-gamma expression. In these studies, we examined the effect of endotoxin or its proximal mediator, tumor necrosis factor (TNF), on the expression of PPAR-gamma in white (WAT) and brown adipose tissue (BAT) CD-1 mice. We report that treatment with endotoxin, but not TNF, transiently decreased PPAR-gamma mRNA levels 4 hr after treatment. However, endotoxin or TNF treatment decreased PPAR-gamma protein levels after 18 hr, which was at a time when LPL mRNA levels were also depressed. These data suggest that decreased PPAR-gamma expression following endotoxin or TNF treatment may contribute to the hyperlipidemia due to decreased expression of LPL, which would impair triglyceride clearance.

L6 ANSWER 6 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 1997:185734 BIOSIS

DN PREV19979484937

TI Differential PPAR-gamma-2 and RXR<sup>alpha</sup>-alpha expression in the differentiating 3T3-L1 adipocyte.

AU Thilliez, Philippe; Baillie, Rebecca; Clarke, Steven D.

CS Univ. Texas, Austin, TX 78712 USA

SO FASEB Journal, (1997) Vol. 11, No. 3, pp. A353.

Meeting Info: Annual Meeting of the Professional Research Scientists on Experimental Biology 97 New Orleans, Louisiana, USA April 6-9, 1997

ISSN: 0892-6638.

DT Conference; Abstract

LA English

L6 ANSWER 7 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 1997:70483 BIOSIS

DN PREV199790369688

TI Terminal differentiation of human liposarcoma cells induced by ligands for peroxisome proliferator-activated receptor gamma and the retinoid X receptor.

AU Tontonoz, Peter (1); Singer, Samuel; Forman, Barry M.; Samat, Pasha; Fletcher, Jonathan A.; Fletcher, Christopher D. M.; Brun, Regina P.; Mueller, Elisabetta; Altick, Soner; Oppenheim, Heather; Evans, Ronald M.; Spiegelman, Bruce M.

CS (1) Gene Expression Lab., The Salk Inst. Biological Studies, La Jolla, CA 92037 USA

SO Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 1, pp. 237-241.

ISSN: 0027-8424.

DT Article

LA English

AB Induction of terminal differentiation represents a promising therapeutic approach to certain human malignancies. The peroxisome proliferator-activated receptor gamma (PPAR-gamma) and the retinoid X receptor alpha (RXR<sup>alpha</sup>) form a heterodimeric complex that functions as a central regulator of adipocyte differentiation. Natural and synthetic ligands for both receptors have been identified. We demonstrate here that PPAR-gamma is expressed at high levels in each of the major histologic types of human liposarcoma. Moreover, primary human liposarcoma cells can be induced to undergo terminal differentiation by treatment with the PPAR-gamma ligand pioglitazone, suggesting that the differentiation block in these cells can be overcome by maximal activation of the PPAR pathway. We further demonstrate that RXR-specific ligands are also potent adipogenic agents in cells expressing the PPAR-gamma/ RXR<sup>alpha</sup> heterodimer, and that simultaneous treatment of liposarcoma cells with both PPAR-gamma- and RXR-specific ligands results in an additive stimulation of differentiation. Liposarcoma cell differentiation is characterized by accumulation of intracellular lipid, induction of adipocyte-specific genes, and withdrawal from the

cell cycle. These results suggest that PPAR-gamma ligands such as thiazolidinediones and RXR-specific retinoids may be useful therapeutic agents for the treatment of liposarcoma.

L6 ANSWER 8 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 1996:364502 BIOSIS

DN PREV1996990886858

TI A human peroxisome-proliferator-activated receptor-gamma is activated by inducers of adipogenesis, including thiazolidinedione drugs.

AU Lamb, Kevin G.; Tugwood, Jonathan D. (1)

CS (1) Res. Toxicol. Section, Zeneca Central Toxicology Lab., Alderley Park, Macclesfield, Cheshire SK10 4TJ UK

SO European Journal of Biochemistry, (1996) Vol. 239, No. 1, pp. 1-7.

ISSN: 0014-2956.

DT Article

LA English

AB We have cloned a human cognate of the mouse peroxisome-proliferator-activated receptor-gamma (hPPAR-gamma) from a human placenta cDNA library. Sequence analysis reveals a high degree of similarity with the mouse receptor and, like other PPAR, hPPAR-gamma forms heterodimers with the retinoid X receptor (RXR<sup>alpha</sup>) (RXR<sup>alpha</sup> - alpha) and binds in vitro to DNA elements containing direct repeats of the sequence TGACCT. In common with mouse PPAR-gamma, hPPAR-gamma is expressed strongly in adipose tissue, but significant levels also are detectable in placenta, lung and ovary. In vitro trans-activation data suggest hPPAR-gamma is only poorly activated by xenobiotic peroxisome proliferators, although certain fatty acids and eicosanoids are potent activators of this receptor. Both mouse and human PPAR-gamma are capable of being activated by thiazolidinedione drugs, although the two receptors appear to differ in their sensitivity to these compounds. Taken together, these data suggest a high degree of structural and functional similarity between mouse and human PPAR-gamma, and provide evidence for variation in human receptor structure which may result in differential sensitivity to activators.

L6 ANSWER 9 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 1995:510753 BIOSIS

DN PREV199598515803

TI cDNA cloning and characterization of the transcriptional activities of the hamster peroxisome proliferator-activated receptor haPPAR-gamma.

AU Aperlo, Christel; Pognonec, Philippe; Saladin, Regis; Auwerx, Johan (1); Boulikos, Kim E.

CS (1) Inst. Pasteur Lille, 1 rue Calmette, 59019 Lille France

SO Gene (Amsterdam), (1995) Vol. 162, No. 2, pp. 297-302.

ISSN: 0378-1119.

DT Article

LA English

AB We have isolated a cDNA corresponding to the hamster peroxisome proliferator-activated receptor haPPAR-gamma, a member of the steroid nuclear hormone receptor superfamily of transcription factors. haPPAR-gamma is highly expressed in adipose tissue, and is expressed in lung, heart, liver and spleen to a lower extent. Thus, haPPAR-gamma may function in activating the transcription of target genes in a variety of tissues, including those not particularly subjected to peroxisomal beta-oxidation. haPPAR-gamma binds efficiently in the presence of retinoid X receptor (RXR<sup>alpha</sup>) (RXR<sup>alpha</sup> - alpha) to a peroxisome proliferator response element (PPRE) first identified in the acyl-CoA oxidase (ACO) promoter, the rate-limiting enzyme of peroxisomal beta-oxidation. The gene (ACO) encoding this enzyme has been previously shown to be under the transcriptional control of mouse PPAR (mPPAR). Although binding of haPPAR-gamma/ RXR<sup>alpha</sup> - alpha on the PPRE of the ACO promoter in vitro is similar to that observed for mPPAR/ RXR<sup>alpha</sup> - alpha, we show that the transcriptional activities of mPPAR and haPPAR-gamma are regulated differently in vivo in response to peroxisome proliferators and heterodimerization with RXR.

L6 ANSWER 10 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 1995:387777 BIOSIS

DN PREV199598402077

TI The combine effect of two transcription factors c/EBP- alpha and RXR-gamma-2 stimulates adipogenesis in fibroblasts.

AU Vasseur-Cognet, Mireille

SO M-S (Medecine Sciences), (1995) Vol. 11, No. 4, pp. 625-626.

ISSN: 0767-0974.

DT Article

LA French

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 NEWS 25 Feb 28 PCTFULL now contains images  
 NEWS 26 Mar 04 SDI PACKAGE for monthly delivery of multiple SDI results  
 NEWS 27 Mar 20 EVENTLINE will be removed from STN  
 NEWS 28 Mar 24 PATDAFULL now available on STN  
 NEWS 29 Mar 24 Additional information for trade-named substances without structures available in REGISTRY  
 NEWS 30 Apr 11 Display format in DGENE enhanced  
 NEWS 31 Apr 14 MEDLINE Reload  
 NEWS 32 Apr 17 Polymer searching in REGISTRY enhanced  
 NEWS 33 Apr 21 Indexing from 1947 to 1956 being added to records in CA/CAPLUS  
 NEWS 34 Apr 21 New current-awareness alert (SDI) frequency in WPIDS/WINDEXNPX  
 NEWS 35 Apr 28 RDISCLOSURE now available on STN  
 NEWS 36 May 05 Pharmacokinetic information and systematic chemical names added to PHARMACOKINETIC  
 NEWS 37 May 15 MEDLINE file segment of TOXCENTER reloaded  
 NEWS 38 May 15 Supporter information for ENCOMPPAT and ENCOMPLIT updated  
 NEWS 39 May 18 CHEMREACT will be removed from STN  
 NEWS 40 May 19 Simultaneous left and right truncation added to WSCA  
 NEWS 41 May 19 RAPRA enhanced with new search field, simultaneous left and right truncation  
 NEWS 42 Jun 08 Simultaneous left and right truncation added to CBNB  
 NEWS 43 Jun 08 PASCAL enhanced with additional data

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L1 324 ADIPO? AND RXR

>> s RXR (3a) alpha  
L2 2709 RXR (3a) ALPHA

>> s adip0?  
L3 125860 ADIPO?

>> s l3 and l2  
L4 133 L3 AND L2

>> dup rem l4  
PROCESSING COMPLETED FOR L4  
L5 77 DUP REM L4 (56 DUPLICATES REMOVED)

>> s l5 and py<=2000  
1 FILES SEARCHED..  
L6 50 L5 AND PY<=2000

>> d bib abs

L6 ANSWER 1 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

AN 2000:335743 BIOSIS

DN PREV200000335743

T1 A PPARgamma mutant serves as a dominant negative inhibitor of PPAR signaling and is localized in the nucleus.

AU Berger, Joel (1); Patel, Hansa V.; Woods, John; Hayes, Nancy S.; Parent, Stephen A.; Clemas, Joseph; Leibowitz, Mark D.; Elbrecht, Alex; Rachubinski, Richard A.; Capone, John P.; Moller, David E.

CS (1) Department of Molecular Endocrinology, Merck Research Laboratories, RY80N-C31, 126 E. Lincoln Avenue, Rahway, NJ, 07065 USA

SO Molecular and Cellular Endocrinology, ( \*\*\*April 25, 2000\*\*\* ) Vol. 162, No. 1-2, pp. 57-67. print.  
ISSN: 0303-7207.

DT Article

LA English

SL English

AB The peroxisomal proliferator-activated receptors (PPARs) are members of the nuclear receptor superfamily that act as ligand-activated transcription factors. PPARgamma plays a critical role in regulating adipocyte differentiation and lipid metabolism. Recently, thiazolidinedione (TZD) and select non-TZD anti-diabetic agents have been identified as PPARgamma agonists. To further characterize this receptor subclass, a mutant hPPARgamma lacking five carboxyl-terminal amino acids was produced (hPPARgamma2DELTA500). In COS-1 cells transfected with PPAR-responsive reporter constructs, the mutant receptor could not be

activated by a potent PPARgamma agonist. When cotransfected with hPPARgamma2 or hPPARalpha, hPPARgamma2DELTA500 abrogated wild-type receptor activity in a dose-responsive manner. hPPARgamma2DELTA500 was also impaired with respect to binding of a high-affinity radioligand. In addition, its conformation was unaffected by normally saturating concentrations of PPARgamma agonist as determined by protease protection experiments. Electrophoretic mobility shift assays demonstrated that hPPARgamma2DELTA500 and hPPARgamma2 both formed heterodimeric complexes with human retinoid X receptor alpha (RXRalpha) and could bind a peroxisome proliferator-responsive element (PPRE) with similar affinity. Therefore, hPPARgamma2DELTA500 appears to repress PPAR activity by competing with wild type receptor to dimerize with RXR and bind the PPRE. In addition, the mutant receptor may titrate out factors required for PPAR-regulated transcriptional activation. Both hPPARgamma2 and hPPARgamma2DELTA500 localized to the nucleus of transiently transfected COS-1 cells as determined by immunofluorescence using a PPARgamma-specific antibody. Thus, nuclear localization of PPARgamma occurs independently of its activation state. The dominant negative mutant, hPPARgamma2DELTA500, may prove useful in further studies to characterize PPAR functions both in vitro and in vivo

>> d bib abs 2

L6 ANSWER 2 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
AN 2000:314239 BIOSIS

DN PREV200000314239

T1 Sterol upregulation of human CETP expression in vitro and in transgenic mice by an LXR element.

AU Luo, Yi; Tai, Alan R. (1)

CS (1) Division of Molecular Medicine, Department of Medicine, Columbia University, New York, NY, 10032 USA

SO Journal of Clinical Investigation, ( \*\*\*February, 2000\*\*\* ) Vol. 105, No. 4, pp. 513-520. print.  
ISSN: 0021-9738.

DT Article

LA English

SL English

AB The cholesterol ester transfer protein (CETP) facilitates the transfer of HDL cholesterol esters from plasma to the liver. Transgenic mice expressing human CETP, controlled by its natural flanking region, increase expression of this gene in response to hypercholesterolemia. We established a CETP promoter-luciferase reporter assay in differentiated 3T3-L1 \*\*\*adipocytes\*\*\* to map the sterol upregulatory element. Promoter mutagenesis suggested that a direct repeat of a nuclear receptor binding sequence separated by 4 nucleotides (DR4 element, -384 to -399) was responsible for this activity. Using mice carrying normal or mutated promoter sequences, we confirmed the importance of this element for gene induction by dietary sterol. A gel retardation complex containing LXR/RXR was identified using the CETP DR4 element and \*\*\*adipocyte\*\*\* nuclear extracts. Both LXRAalpha/RXRalpha and LXRBeta/RXRalpha transactivated the CETP promoter via its DR4 element in a sterol-responsive fashion. Thus, the positive sterol response of the CETP gene is mediated by a nuclear receptor binding site that is activated by LXR. That Cyp7a, the rate-limiting enzyme for conversion of cholesterol into bile acids in the liver, is also regulated by LXRAalpha suggests that this class of nuclear receptor coordinates the regulation of HDL cholesterol ester catabolism and bile acid synthesis in the liver.

>> d bib abs 3

L6 ANSWER 3 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
AN 1998:492788 BIOSIS

DN PREV199800492788

T1 A novel 3T3-L1 preadipocyte variant that expresses PPARgamma2 and RXRalpha but does not undergo differentiation.

AU Baillie, Rebecca A.; Sha, Xiaoming; Thuillier, Philippe; Clarke, Steven D.

(1)

CS (1) Inst. Mol. Biol., Univ. Tex, Austin, TX 78712 USA

SO Journal of Lipid Research, ( \*\*\*Oct., 1998\*\*\* ) Vol. 39, No. 10, pp. 2046-2053.  
ISSN: 0022-2275.

DT Article

LA English

AB This report describes a novel \*\*\*adipocyte\*\*\*-like cell line termed 3T3-L1/RB1 that was derived from preadipocyte cell line, 3T3-L1. The 3T3-L1/RB1 cells continued to divide after reaching confluence, formed foci, and constitutively expressed a low level of \*\*\*adipose\*\*\* fatty acid binding protein (A-FABP) mRNA. However, 3T3-L1/RB1 cells did not undergo terminal differentiation as indicated by the failure of insulin and thiazolidinediones to induce the expression of A-FABP, lipoprotein lipase, and fatty acid synthase. We hypothesized that the 3T3-L1/RB1 variant did not respond to differentiation stimuli because it did not express either peroxisomal proliferator-activated receptor gamma2 (PPARgamma2) or its heterodimer partner, retinoid X receptor alpha (RXRalpha). Surprisingly, Western blots revealed that 3T3-L1/RB1 cells contained both PPARgamma2 and RXRalpha proteins at levels equal to or greater than that of the parent cell line. However, gel retardation assays using the \*\*\*adipose\*\*\* response element from A-FABP and nuclear protein extracts from 3T3-L1/RB1 cells treated with insulin or pioglitazone revealed that nuclear protein extracts from 3T3-L1/RB1 cells had very little ability to bind the PPARgamma2 recognition sequence of the A-FABP gene. These data suggest that the 3T3-L1/RB1 variant contains a mutation that may prevent ligand activation of PPARgamma2, and the subsequent conversion of 3T3-L1/RB1 cells to mature fat cells.

>> d bib abs 4-10

L6 ANSWER 4 OF 50 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.  
AN 1997:366218 BIOSIS

DN PREV19970068151

T1 Recent update on the PPAR-alpha-null mouse.

AU Gonzalez, F. J.

CS Lab. Metabolism, Div. Basic Sci., Natl. Cancer Inst., Build. 37, Room 3E-24, NIH, Bethesda, MD 20892 USA

SO Biochimie (Paris), (1997) Vol. 79, No. 2-3, pp. 139-144.  
ISSN: 0300-9084.

DT Journal: Article

LA English

AB Short-term treatment of rats and mice with peroxisome proliferators (PP)